The Decline in Blood Lead Levels in the United States

The National Health and Nutrition Examination Surveys (NHANES)

James L. Pirkle, MD. PhD; Debra J. Brody, MPH; Elaine W. Gunter; Rachel A. Kramer, ScD; Daniel C. Paschal, PhD; Katherine M. Flegal, PhD; MPH; Thomas D. Matte, MD; MPH; **

Objective.—To describe trends in blood lead levels for the US population and selected population subgroups during the time period between 1976 and 1991.

Design.—Two nationally representative cross-sectional surveys and one cross-sectional survey representing Mexican Americans in the southwestern United States.

Setting/Participants.—Participants in two national surveys that included blood lead measurements: the second National Health and Nutrition Examination Survey, 1976 to 1980 (n=9832), and phase 1 of the third National Health and Nutrition Examination Survey, 1988 to 1991 (n=12119). Also, Mexican Americans participating in the Hispanic Health and Nutrition Examination Survey, 1982 to 1984 (n=5682).

Results.—The mean blood lead level of persons aged 1 to 74 years dropped 78%, from 0.62 to 0.14 µmol/L (12.8 to 2.8 µg/dL). Mean blood lead levels of children aged 1 to 5 years declined 77% (0.66 to 0.15 µmol/L [13.7 to 3.2 µg/dL]) for non-Hispanic white children and 72% (0.97 to 0.27 µmol/L [20.2 to 5.6 µg/dL]) for non-Hispanic black children. The prevalence of blood lead levels 0.48 µmol/L (10 µg/dL) or greater for children aged 1 to 5 years declined from 85.0% to 5.5% for non-Hispanic white children and from 97.7% to 20.6% for non-Hispanic black children. Similar declines were found in population subgroups defined by age, sex, race/ethnicity, income level, and urban status. Mexican Americans also showed similar declines in blood lead levels of a slightly smaller magnitude over a shorter time.

Conclusions.—The results demonstrate a substantial decline in blood lead levels of the entire US population and within selected subgroups of the population. The major cause of the observed decline in blood lead levels is most likely the removal of 99.8% of lead from gasoline and the removal of lead from soldered cans. Although these data indicate major progress in reducing lead exposure, they also show that the same sociodemographic factors continue to be associated with higher blood lead levels, including younger age, male sex, non-Hispanic black race/ethnicity, and low income level. Future efforts to remove other lead sources (eg, paint, dust, and soil) are needed but will be more difficult than removing lead from gasoline and soldered cans.

(JAMA. 1994;272:284-291)

LEAD has been dispersed in the environment in substantial quantities over a long period of time. Compelling evidence from the scientific community on a wide range of adverse health outcomes has placed lead in the forefront of environmental health concerns. In the 1970s. federal regulatory and legislative efforts were undertaken to reduce lead hazards, including actions to limit the use of lead in paint and gasoline.1 The second National Health and Nutrition Examination Survey (NHANES II, 1976) to 1980) established baseline lead measurements for the US population and demonstrated the pervasiveness of lead

See also pp 277 and 315.

exposure across race, urban and rural residence, and income levels.² Data from NHANES II showed a decline in blood lead levels from the beginning to the end of the survey period that was closely correlated to declines in the use of leaded gasoline during these years.³

Since 1980, intensive federal, state, and local actions directed at primary prevention have been taken to further reduce lead exposure from gasoline, paint, solder, and other sources. Secondary prevention activities, such as screening for early detection and lead education programs, have also been implemented. New data from phase 1 of the third National Health and Nutrition Examination Survey (NHANES III phase 1, 1988 to 1991) permit examination of changes in blood lead levels since 1980 in the US population and evaluation of the impact of these regulatory

From the Division of Environmental Health Laboratory Sciences (Drs Pirkle and Paschal and Ms Gunter) and Lead Poisoning Prevention Branch (Dr Matte), National Center for Environmental Health, Centers for Disease Control and Prevention, Altanta, Ga, and Division of Health Examination Statistics, National Center for Health Statistics, Centers for Disease Control and

Prevention, Hyattsville, Md (Drs Kramer and Flegal and Ms Brody).

Reprint requests to Division of Environmental Health Laboratory Sciences. National Center for Environmental Health, Centers for Disease Control and Prevention. MS-F20, Atlanta. GA 30333 (Dr Pirkle).

actions. The Hispanic Health and Nutrition Examination Survey (HHANES, 1982 to 1984) provides data on Mexican Americans at an intermediate time point. These analyses of trends in blood lead levels serve both to evaluate the effectiveness of prevention programs and to develop new strategies to further reduce lead exposure in the United States.

METHODS

Design and Data Collection

The National Health and Nutrition Examination Surveys (NHANES) are designed to measure and monitor the health and nutritional status of the US population. The general design of the NHANES is a stratified multistage probability cluster sample of households whose target population is civilian noninstitutionalized persons residing in the United States. Blood lead levels were determined in NHANES II (1976 to 1980), HHANES (1982 to 1984), and NHANES III (1988 to 1994). The estimates from NHANES II and NHANES III are based on a national sample, whereas HHANES sampled three Hispanic subgroups.46

National trends of blood lead levels presented in this article were based primarily on comparisons of data from NHANES II and NHANES III phase 1 (1988 to 1991). Trends for Mexican Americans were based on a comparison between the estimates from HHANES and NHANES III phase 1. The HHANES also provides an intermediate point in time between NHANES II and NHANES III. The HHANES sample of Mexican Americans included only those residing in the southwestern United States whereas the NHANES III phase I sample represented Mexican Americans residing in the entire United States.

Venous blood lead measurements were obtained for persons aged 6 months to 74 years in NHANES II; persons aged 4 to 74 years in HHANES; and persons aged 1 year and older in NHANES III phase 1. Analysis was limited to persons aged 1 to 74 years for national trends and aged 4 to 74 years for trends in the Mexican-American population. The final samples used for analyses included 9832 and 12119 for the national trends from NHANES II and NHANES III phase 1, respectively, and 5682 and 4067 Mexican Americans from HHANES and NHANES III phase 1, respectively. Data from all of the surveys were collected using a household interview followed by a detailed medical examination in a mobile examination center.

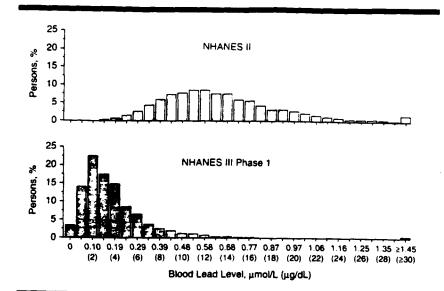


Fig 1.—Blood lead levels for persons aged 1 to 74 years: United States, second National Health and Nutrition Examination Survey (1976 to 1980, top) and phase 1 of the third National Health and Nutrition Examination Survey (1988 to 1991, bottom).

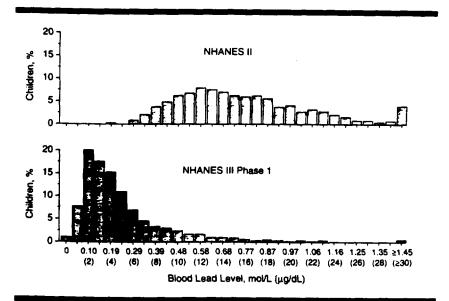


Fig 2.—Blood lead levels for children aged 1 to 5 years: United States, second National Health and Nutrition Examination Survey (1976 to 1980, top) and phase 1 of the third National Health and Nutrition Examination Survey (1988 to 1991, bottom).

The response rates for blood lead collection in the three surveys ranged from 61% to 69%. Previous nonresponse bias analyses conducted for NHANES II, HHANES, and NHANES III phase 1 indicated that there was no apparent bias due to nonresponse.⁷⁻⁹

Laboratory Methods

All venous blood specimens were collected in the mobile examination centers, frozen, and shipped on dry ice to the NHANES laboratory, Division of Environmental Health Laboratory Sciences, National Centers for Environmental Health, Centers for Disease Control and Prevention, Atlanta, Ga, for analysis. The methods for determining lead in blood, including descriptions of quality control and assurance procedures, have been described for each survey. P11 Comparability has been established for the method used in NHANES II and HHANES (modified Delves cup) and that used in NHANES III phase 1 (graphite furnace atomic absorption spectrophotometry), as described by Miller et al. 12 In each of the three sur-

Table 1 —Distribution of Blood Lead Levels for Persons Aged 1 to 74 Years by Age Category. Sex. Race Ethnicity. Urban Status, and income Level United States 1976 to 1980 (Second National Health and Nutrition Examination Survey (NHANES II)) and 1988 to 1991 (Phase 1 of the Third National Health and Nutrition Examination Survey [NHANES III Phase 1]1

			95% Confidence	Percentiles, µmol/L (µg/dL)						
	No.	Geometric Mean, µmol/L (µg/dL)*	interval, µmol/L (µg/dL)	5th	10th	25th	50th	75th	90th	95th
All persons 1976-1980	9832	0.62 (12.8)	0.60-0.65 (12.4-13.3)	0.34 (7.0)	0.39 (8.0)	0.48 (10.0)	0.63 (13.0)	0.82 (17.0)	1.01 (21.0)	1.21 (25.0
1988-1991	12119	0.14 (2.8)	0.13-0.15 (2.7-3.0)	<0.05 (<1.0)	0.05	0.09	0.14 (3.0)	0.23	0.35 (7.3)	0.45
Ages 1-5 y 1976-1980	2271	0.71 (15.0)	0.67-0.75 (14.2-15.8)	0.39 (8.0)	0.43 (9.0)	0.58	0.72	0.92	1.16 (24.0)	1.35
1988-1991	2234	0.17 (3.6)	0.16-0.19 (3.3-4.0)	0.05	0.07 (1.5)	(12.0) 0.11 (2.2)	(15.0) 0.18 (3.7)	(19.0) 0.28 (5.9)	0.46 (9.6)	0.59 (12.2
Ages 6-19 y 1976-1980	2024	0.56 (11.7)	0.54-0.60 (11.2-12.4)	0.29 (6.0)	0.34 (7.0)	0.43 (9.0)	0.58 (12.0)	0.72 (15.0)	0.92 (19.0)	1.06
1988-1991	2963	0.09	0.08-0.11 (1.7-2.2)	<0.05 (<1.0)	<0.05 (<1.0)	0.06	0.10 (2.1)	0.17 (3.5)	0.26 (5.4)	0.36
Ages 20-74 y 1976-1980	5537	0.63	0.61-0.66	0.34 (7.0)	0.39	0.48	0.63	0.82	1.06	1.25
1988-1991	6922	(13.1) 0.14 (3.0)	(12.7-13.7) 0.14-0.15 (2.8-3.2)	<0.05 (<1.0)	0.06 (1.2)	(10.0) 0.10 (2.0)	(13.0) 0.15 (3.2)	(17.0) 0.24 (5.0)	(22.0) 0.36 (7.4)	0.46 (9.5
Males	4005	0.72	0.70-0.75	0.39	0.43	0.58	0.72			1.30
1976-1980	4895	(15.0)	(14.5-15.5)	(8.0)	(9.0)	(12.0)	(15.0)	0.92 (19.0)	1.16 (24.0)	(27.0
1988-1991	6051	0.18 (3.7)	0.17-0.19 (3.5-3.9)	0.06 (1.2)	0.08	0.12 (2.4)	0.18 (3.8)	0.28 (5.8)	0.42 (8.7)	0.52 (10.9
Females 1976-1980	4937	0.54 (11.1)	0.51-0.55 (10.6-11.5)	0.29 (6.0)	0.34 (7.0)	0.43 (9.0)	0.53 (11.0)	0.68 (14.0)	0.87 (18.0)	0.97 (20.0
1988-1991	6068	0.10 (2.1)	0.10-0.11 (2.0-2.2)	<0.05 (<1.0)	<0.05 (<1.0)	0.07 (1.4)	0.11 (2.3)	0.18 (3.8)	0.28 (5.7)	0.36 (7.4
Non-Hispanic whites 1976-1980	6816	0.61 (12.6)	0.58-0.63 (12.1-13.1)	0.29 (6.0)	0.39 (8.0)	0.48 (10.0)	0. 63 (13.0)	0.77 (16.0)	1.01 (21.0)	1.16
1988-1991	4337	0.13 (2.7)	0.12-0.14 (2.2-2.8)	<0.05 (<1.0)	0.05 (1.0)	0.06 (1.7)	0.14 (2.9)	0.22 (4.5)	0.33 (6.8)	0.43 (8.9
Non-Hispanic blacks 1976-1980	1259	0.70 (14.5)	0.66-0.74 (13.7-15.5)	0.39 (8.0)	0.43 (9.0)	0.53 (11.0)	0.72 (15.0)	0.92 (19.0)	1.11 (23.0)	1.30
1988-1991	3274	0.17 (3.5)	0.16-0.19 (3.3-3.9)	<0.05 (<1.0)	0.06 (1.3)	0.11 (2.2)	0.18 (3.7)	0.28 (5.9)	0.45 (9.3)	0.58 (12.1
Non-central city 1976-1980	7112	0.60 (12.5)	0.58-0.64 (12.0-13.1)	0.29 (6.0)	0.34 (7.0)	0.48 (10.0)	0.63 (13.0)	0.77 (16.0)	1.01 (21.0)	1.16
1988-1991	7495	0.13 (2.7)	0.12-0.14 (2.5-2.8)	<0.05 (<1.0)	0.05 (1.0)	0.09 (1.8)	0.14 (3.0)	0.22 (4.6)	0.33 (8.9)	0.43
Central city, <1 million 1976-1980	1612	0.66 (13.6)	0.61-0.70 (12.7-14.5)	0.34 (7.0)	0.39	0.53 (11.0)	0.68 (14.0)	0.87 (18.0)	1.08 (22.0)	1.25
1988-1991	2909	0.14 (2.9)	0.12-0.16 (2.5-3.4)	<0.05 (<1.0)	0.05	0.09	0.14 (3.0)	0.25 (5.2)	0.40 (8.3)	0.50
Central city, ≥1 million 1976-1980	1108	0.67 (13.9)	0.61-0.73 (12.7-15.1)	0.34 (7.0)	0.43 (9.0)	0.53 (11.0)	0.68 (14.0)	0.87 (18.0)	1.08 (22.0)	1.21
1988-1991	1379	0.19 (3.9)	0.17-0.21 (3.6-4.3)	0.06 (1.3)	0.09	0.12 (2.5)	0.19 (4.0)	0.29 (6.1)	0.48 (9.9)	0.64
Income level, low† 1976-1980	2548	0. 63 (13.1)	0.60-0.67 (12.4-13.8)	0.29 (6.0)	0.34 (7.0)	0. 48 (10.0)	0. 63 (13.0)	0.82 (17.0)	1.11 (23.0)	1.25
1988-1991	4108	0.16 (3.4)	0.15-0.18 (3.1-3.8)	<0.05 (<1.0)	0.06 (1.3)	0.10 (2.1)	0.17 (3.6)	0.28 (5.8)	0.45 (9.4)	0.5
ncome level, mid† 1976-1980	4176	0.61 (12.6)	0.58-0.63 (12.1-13.1)	0.34 (7.0)	0.39 (8.0)	0.48 (10.0)	0. 63 (13.0)	0.77 (16.0)	1.01 (21.0)	1.10
1988-1991	4050	0.13 (2.7)	0.13-0.14 (2.6-2.9)	<0.05 (<1.0)	0.05	0.08 (1.7)	0.14 (2.9)	0.23	0.34 (7.1)	0.44
ncome level, hight 1976-1980	2784	0.63 (13.0)	0.80-0.65 (12.5-13.5)	0.34 (7.0)	0.39 (8.0)	0.48 (10.0)	0.63 (13.0)	0.82	1.01 (21.0)	1.2
1988-1991	2781	0,12 (2.5)	0.12-0.13 (2.4-2.7)	<0.05 (<1.0)	<0.05 (<1.0)	0.08 (1.7)	0.14 (2.8)	0.21 (4.3)	0.30 (6.3)	0.39 (8.0

^{*}For each grouping, the geometric means from NHANES II and NHANES III phase 1 are statistically different (P<.01), 1 throome level was defined by poverty-income ratio (PIR) categorized as low (0<PIR<1.30), mid (1.30≤PIR<3.00), and high (PIR≥3.00).

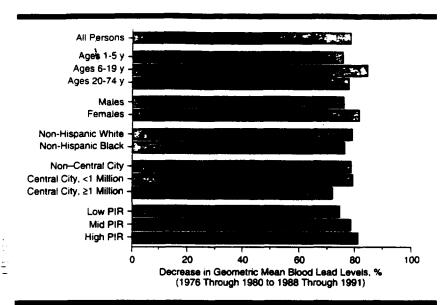


Fig 3.—Percentage decrease in geometric mean blood lead levels for persons aged 1 to 74 years by age category, sex, race/ethnicity, urban status, and income level: United States, 1976 to 1980 (second National Health and Nutrition Examination Survey) to 1988 to 1991 (phase 1 of the third National Health and Nutrition Examination Survey). Income level defined by poverty-income ratio (PIR) as low (0<PIR<1.30), mid (1.30≤PIR<3.00), and high (PIR≥3.00).

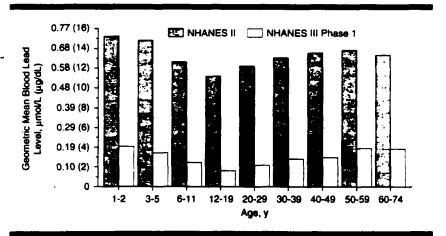


Fig 4.—Geometric mean blood lead levels for persons aged 1 to 74 years by age: United States, 1976 to 1980 (second National Health and Nutrition Examination Survey [NHANES II]) and 1988 to 1991 (phase 1 of the third National Health and Nutrition Examination Survey (NHANES III phase 1]).

veys, the blood lead measurements were calibrated using standards prepared from lead nitrate Standard Reference Material 928 obtained from the National Institute of Standards and Technology, Gaithersburg, Md. The consistent use of Standard Reference Material 928 for calibration assured a common accuracy base across surveys.

Demographic and Socioeconomic Covariates

The trends analysis included stratification by five sociodemographic variables: age, sex, race/ethnicity, urban status, and income level. Age was defined in years and categorized as 1 to 5 years (4 to 5 years for Mexican Americans), 6 to 19 years, and 20 to 74 years for analysis. Race/ethnicity was categorized as non-Hispanic black, non-Hispanic white, and Mexican American. Because of small sample sizes, persons not defined by these three largest US race/ethnicity groups were included only in the overall estimates.

Definitions for income level and urban status were based on those previously determined by the US Bureau of the Census. Income level was defined by the poverty-income ratio (PIR): the total family income divided by a poverty threshold. The PIR was divided into three categories: low (0<PIR<1.30), mid $(1.30 \le PIR < 3.00)$, and high $(PIR \ge 3.00)$ Urban status was categorized as noncentral city, central city with population less than I million, and central city with population 1 million or greater.

Statistical Analysis

Survey-specific sample weights were used in all statistical analyses. Geometric means and percentiles of blood lead were calculated using SAS.13 Log10 transformed blood lead levels were used to normalize the distribution of blood lead levels. Geometric means were calculated by taking the antilog of the mean log10 blood lead levels. SUDAAN,14 a statistical software package that incorporates the sample weights and adjusts for the complex sample design of the survey, was used to estimate the SEs.

RESULTS

The results of the trend analysis in blood lead levels are presented in two parts: first, the change in blood lead levels from NHANES II (1976 to 1980) to NHANES III phase 1 (1988 to 1991), and second, the change in blood lead levels for Mexican Americans from HHANES (1982 to 1984) to NHANES III phase 1 (1988 to 1991).

From NHANES II (1976 To 1980) to NHANES # Phase 1 (1988 To 1991)

The different distributions of blood lead levels for those aged 1 to 74 years from NHANES II and NHANES III phase 1 are presented in Fig 1. A decline of approximately 0.48 µmol/L (10 µg/ dL) occurred in the geometric mean blood lead level as well as a clear change in the shape of the distribution. When the sample was limited to children aged 1 to 5 years, similar results were observed (Fig 2).

The geometric means, 95% confidence intervals, and percentiles of the blood lead distribution for the total population and stratified by the five sociodemographic factors are presented by survey in Table 1. For the total population, the geometric mean decreased by 0.48 μmol/L (10 μg/dL). Stratification of the data showed that the size of the decrease was fairly constant across sex, race/ethnicity, age groups, urban sta-

tus, and income levels. The decline represents an overall de-

crease in blood lead levels of 78% for persons aged 1 to 74 years and a decrease of 70% or more for selected subgroups (Fig 3). Children and youths aged 6 to 19 years showed the greatest decline in blood lead levels. How-

ever, a decline of 0.48 µmol/L (10 µg/

Table 2 A Provincial Teams of the 14 Years at or Above Selected Blood Level Outoffs by Age. Sex. Race Ethnich, Turban Status, and income Level On ted States, 1976 to 1980 (Second National Health and Nutrition Examination Survey) and 1988 to 1991 (Phase 1 of the Third National Health and Nutrition Examination Survey)

	No.	Blood Lead Levels of Population Group, %							
_		≥1.45 µmol/L (≥30 µg/dL)	≥1.21 µmol/L (≥25 µg/dL)	≥0.97 µmoVL (≥20 µg/dL)	≥0.72 µmoVL (≥15 µg/dL)	≥0.48 µmoi/L (≥10 µg/dL)	≥0.24 µmoV\ (≥5 µ g/dL)		
All persons 1976-1980	9832	1.9	5.2	14.9	37.7	77.8	99.2		
1988-1991	12 119	0.2	0.4	0. 6	1.1	4.3	23.3.		
Ages 1-5 y 1976-1980	2271	4.1	9.3	24.7	52.6	88.2	99.8		
1988-1991	2234	0.4	0.5	1.1	2.7	8.9	33.2		
Ages 6-19 y 1976-1980	2024	0.6	2.4	8.2	27.7	71.7	99.1		
1988-1991	2963	0.0	0.2	0.4	0.8	2.6	12.2		
Ages 20-74 y 1976-1980	5537	2.3	5.9	16.7	40.3	79.4	99.2		
1988-1991	6922	0.3	0.4	0.7	1.1	4.4	25.5		
Males 1976-1980	4895	3.3	9.0	24.1	53.1	89.6	99.8		
1988-1991	6051	0.4	0.7	1.1	1.9	6.8	33.5		
Females 1976-1980	4937	0.6	1.6	6.2	23.0	66.7	98.7		
1988-1991	6068	0.1	0.1	0.2	0.4	1.8	13.2		
Non-Hispanic whites 1976-1980	6816	1.7	4.8	14.0	36.0	76.9	99.2		
1988-1991	4337	0.2	0.4	0.6	0.9	3.6	, 21.1		
Non-Hispanic blacks 1976-1980	1259	2.8	8.4	22.9	50.9	86.4	99.7		
1988-1991	3274	0.2	0.4	1.2	2.6	8.5	33.7		
Non-central city 1976-1980	7112	1.9	4.9	13.9	35.3	75.7	99.0		
1988-1991	7495	0.1	0.3	0.6	0.9	3.5	21.7		
Central city, <1 million 1976-1980	1612	1.9	6.1	17.1	43.1	82.1	99.8		
1988-1991	2909	0.3	0.3	0.6	1.8	5.9	26.9		
Central city, ≥1 million 1976-1980	1108	1.8	6.0	18.4	44.4	84.8	99.9		
1988-1991	1379	1.1	1,4	1.9	2.9	9.8	36.0		
income level, low* 1976-1980	2548	2.9	6.8	18.0	39.6	78.4	99.2		
1988-1991	4106	0.5	0.9	1.8	2,6	8.8	32.6		
Income level, mid* 1976-1980	4176	1.7	4.6	13.8	36.3	76.1	99.3		
1988-1991	4050	0.2	0.3	0.5	0.9	3.4	22.9		
Income level, high* 1976-1980	2784	1.5	5.1	14.5	38.3	79.8	99.4		
1988-1991	2781	0.1	0.3	0.4	0.6	2.7	18.4		

^{*}Income level was defined by poverty-income ratio (PIR) categorized as low (0<PIR<1.30), mid (1.30≤PIR<3.00), and high (PIR≥3.00).

dL) or greater between NHANES II and NHANES III phase 1 was consistent across the entire age range (Fig 4).

The percentage of the population with blood lead levels at or above selected values is presented in Table 2. These levels were chosen in part because of their prior or potential use in public health policy. For those aged 1 to 74 years, the prevalence of blood lead levels 0.48 µmol/L (10 µg/dL) or greater decreased from 77.8% in NHANES II to 4.3% in NHANES III phase 1. For children aged 1 to 5 years during the same time frame, the prevalence of blood lead levels 0.48 µmol/L (10 µg/dL) or greater decreased from 88.2% to 8.9%.

The change in percentage of children at or above selected lead levels from NHANES II to NHANES III phase 1 is presented in Fig 5.

Separate analysis by race/ethnicity revealed that geometric mean blood lead levels declined by 77%, from 0.66 to 0.15 µmol/L (13.7 to 3.2 µg/dL), for non-Hispanic white children and by 72%, from 0.97 to 0.27 µmol/L (20.2 to 5.6 µg/dL), for non-Hispanic black children. The prevalence of blood lead levels 0.48 µmol/L (10 µg/dL) or greater for children in this same age group declined from 85.0% to 5.5% for non-Hispanic white children and from 97.7% to 20.6% for non-Hispanic black children.

Mean blood lead levels decreased from

0.73 to 0.20 μ mol/L (15.2 to 4.1 μ g/dL) for children aged 1 to 2 years and from 0.71 to 0.16 μ mol/L (14.8 to 3.4 μ g/dL) for children aged 3 to 5 years. During the same time period, the prevalence of blood lead levels 0.48 μ mol/L (10 μ g/dL) or greater also decreased from 88.3% to 11.5% for children aged 1 to 2 years and from 88.1% to 7.3% for children aged 3 to 5 years.

Mean blood lead levels decreased by 60% (1.16 to 0.47 µmol/L [24.0 to 9.7 µg/dL]) for non-Hispanic black children from low-income families living in the central cities with populations of 1 million or more. This compares with an overall decrease in blood lead levels of 75% (0.72 to 0.18 µmol/L [14.9]

to 3.6 μ g/dL]) for all children aged 1 to 5 years.

From HHANES (1982 to 1984) to NHANES III Phase 1 (1988 to 1991)

The HHANES was conducted from 1982 to 1984, between the second and third NHANES. Geometric mean blood lead levels were also found to be intermediate between the estimates of the two national surveys. The blood lead levels of Mexican Americans from HHANES were lower than overall levels observed in NHANES II, but not as low as levels of Mexican Americans sampled in NHANES III phase 1.

Geometric means, 95% confidence intervals, and percentiles of the blood lead distribution of Mexican Americans between HHANES and NHANES III phase 1 are presented in Table 3. Mexican Americans showed an overall decrease in geometric mean of 65%, from 0.41 to 0.14 µmol/L (8.5 to 3.0 µg/dL). The geometric mean for children aged 4 to 5 years declined from 0.52 to 0.17 μmol/L (10.9 to 3.5 μg/dL). As demonstrated in the comparison of NHANES II to NHANES III phase 1 estimates, the size of the decrease in blood lead levels was similar in both sexes and across age groups and income levels.

The proportion of the Mexican-American population at or above selected blood lead levels is shown in Table 4. Overall, prevalence of blood lead levels 0.48 µmol/L (10 µg/dL) or greater among Mexican Americans decreased from 41.2% to 5.9%. The percentage of children aged 4 to 5 years with blood lead levels 0.48 µmol/L (10 µg/dL) or greater decreased from 61.5% to 4.9%. These results demonstrate that one in 20 Mexican Americans aged 4 to 5 years continue to have blood lead levels of health concern.

COMMENT

The data from two national surveys of the US population, conducted more than a decade apart, demonstrate a substantial decline in blood lead levels. As the consequence of a shift in the overall distribution of lead levels, fewer persons have blood lead levels in the upper ranges. The decrease in mean blood lead levels was observed for the total population and within all race/ethnicity, sex, urban status, and income level subgroups examined in this article. The prevalence of blood lead levels 0.48 µmol/L (10 µg/dL) or greater also decreased sharply from 77.8% to 4.3%.

As discussed herein, exposure to lead from major population-wide lead sources declined between 1976 and 1991. Consistent with this decline, the blood lead levels observed in HHANES (1982 to

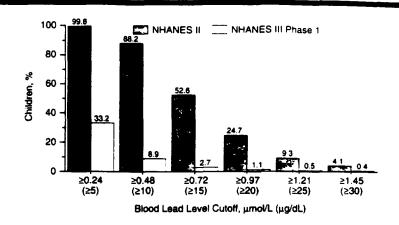


Fig 5.—Percentage of children aged 1 to 5 years at or above selected blood lead levels: United States, 1976 to 1980 (second National Health and Nutrition Examination Survey [NHANES II]) and 1988 to 1991 (phase 1 of the third National Health and Nutrition Examination Survey [NHANES III] phase 1]).

1984) were intermediate between levels found in NHANES II (1976 to 1980) and NHANES III phase 1 (1988 to 1991). Consequently, the magnitude of decrease from HHANES to NHANES III phase 1 (65%) was less than from NHANES II to NHANES III phase 1 (78%). The percentage of Mexican Americans with blood lead levels 0.48 μmol/L (10 μg/dL) or greater declined from 41.2% to 5.9%. In NHANES III phase 1, both mean blood lead levels and the prevalence of blood lead levels 0.48 μmol/L (10 μg/dL) or greater of Mexican Americans were closer to those of non-Hispanic whites than to those of non-Hispanic blacks.

The decline in blood lead levels seen in these national surveys is consistent with the results of other studies of environmental lead levels,1 which indicate that a continued reduction in exposure to lead sources began in the late 1970s and continued throughout the 1980s. Between 1976 and 1991, the three major sources of lead exposure common to the general population were lead in gasoline, soldered cans, and paint. In 1976, a total of 186.47 million kg (205810 tons) of lead was used in gasoline in the United States.15 In 1983, this amount had dropped to 51.59 million kg (56 940 tons), and in 1990, lead used in gasoline had been reduced to 0.47 million kg (520 tons).15 From 1976 to 1990, the amount of lead used in gasoline decreased 99.8%. The reduction of lead in gasoline is most likely the greatest contributor to the observed decline in blood lead levels during the period of the national surveys. 1.3,16,17

Lead from gasoline and soldered cans contribute to lead in food. Since gasoline lead enters food through multiple pathways, 1,18,16 it is difficult to make a quantitative estimate of the reduction in food lead that resulted from decreasing lead in gasoline. The amount of lead used in soldered cans decreased markedly throughout the 1980s. In 1980, 47% of food and soft drink cans were lead soldered. By 1985, this figure had dropped to 14%, and by 1990, only 0.85% of food and soft drink cans were lead soldered. As of November 1991, lead-soldered food or soft drink cans were no longer manufactured in the United States. 18

The Food and Drug Administration uses "market-basket" surveys to estimate the average daily intake of lead from food for various population groups in the United States.19 For 2-year-old children, these surveys estimate the typical daily intake of lead to have dropped from 30 µg/d in 1982 to 1.9 µg/d in 1991. 19,20 The Environmental Protection Agency estimated in 1986 that about 42% of lead in food came from lead-soldered cans. Thus, reducing the amount of lead used in soldered cans has likely been a major factor in reducing food lead levels. Although it is difficult to quantitatively determine the decrease in blood lead levels attributable to reduced amounts of lead in soldered cans. the decline in the amount of lead used in this source probably contributed substantively to the observed decline in blood lead levels.

The manufacture of lead-based paint was limited to less than 0.06% by weight in 1978 by the Consumer Product Safety Commission.¹ Individuals who have left housing with lead-based paint or who reside in lead-abated homes have reduced their lead exposure. Still, lead-based paint remains a problem, predominantly in older, deteriorating housing. 1.15.16 The NHANES do not specifically target persons who live in such

Table 3 —Distribution of Blood Lead Levels for Mexican Americans Aged 4 to 74 Years by Age Category. Sex: and income Level (1982 to 1984) Hisbanic Health and Nutrition Examination Survey (HHANES)) and 1988 to 1991 (Phase 1 of the Third National Health and Nutrition Examination Survey (NHANES) ill phase 1 of the Third National Health and Nutrition Examination Survey (NHANES).

	No.		95% Confidence Interval, µmol/L (µg/dL)	Percentiles, µmol/L (µg/dL)						
		Geometric Mean, µmol/L (µg/dL)*		5th	10th	25th	50th	75th	90th	95th
All persons										
1982-1984	5682	0.41 (8.5)	0.40-0.42 (8.3-8.7)	0.19 (4.0)	0.24 (5.0)	0.29 (6.0)	0.43 (9.0)	0.58 (12.0)	0. <i>77</i> (16.0)	0.87 (18.0)
1988-1991	3611	Q.14 (3.0)	0.12-0.17 (2.5-3.5)	<0.05 (<1.0)	0.05	0.09	0.16 (3.3)	0.26 (5.4)	0.40 (8.3)	0.51 (10.6
Ann. 4.5		(3.0)	(2.5 5.5)	(<1.0)	(1.1)	(1.3)	(0.0)	(5.4)	(0.0)	(10.0
Ages 4-5 y 1982-1984	269	0. 53 (10.9)	0.50-0.56 (10.3-11.5)	0.24 (5.0)	0.29 (6.0)	0.39 (8.0)	0. 53 (11.0)	0. 68 (14.0)	0.92 (19.0)	1.11 (23.0
1988-1991	349	0.17 (3.5)	0.14-0.21 (2.8-4.3)	<0.05 (<1.0)	0.07 (1.4)	0.12 (2.5)	0.18 (3.8)	0.28 (5.9)	0.40 (8.3)	0.48 (9.9)
Ages 6-19 y									·	
1962-1964	2331	0.3 9 (8.0)	0.38-0.40 (7.8-8.2)	0.19 (4.0)	0.24 (5.0)	0.29 (6.0)	0.39 (8.0)	0.53 (11.0)	0.68 (14.0)	0. 82 (17.0)
1988-1991	1188	0.12 (2.5)	0.10-0.15 (2.0-3.2)	<0.05 (<1.0)	<0.05 (<1.0)	0.08 (1.6)	0.14 (2.8)	0.23 (4.7)	0.36 (7.4)	0.47 (9.8)
Ages 20-74 y										
1982-1984	3082	0.42 (5.7)	0.40-0.43 (8.3-9.0)	0.19 (4.0)	0.24 (5.0)	0.29 (6.0)	0.43 (9.0)	0. 58 (12.0)	0.77 (16.0)	0.92 (19.0)
1988-1991	2074	0.15 (3.2)	0.13-0.18 (2.7-3.7)	<0.05 (<1.0)	0.05 (1.1)	0.10 (2.0)	0.16 (3.4)	0.28 (5.7)	0.42 (8.6)	0.54 (11.1)
Males										
1982-1984	2638	0.50 (10.4)	0.4 9 -0.51 (10.2-10.5)	0.24 (5.0)	0.29 (6.0)	0.39 (8.0)	0.48 (10.0)	0. 68 (14.0)	0.87 (18.0)	1.01 (21.0)
1968-1991	1797	0.19 (4.0)	0.16-0.23 (3.3-4.8)	0.0 6 (1.2)	0.0 6 (1.6)	0.12 (2.5)	0.20 (4.2)	0.31 (6.5)	0.45 (9.4)	0.57 (11.8)
Females										
1962-1964	3044	0.34 (7.0)	0.32-0.35 (6.7-7.2)	0.14 (3.0)	0.19 (4.0)	0.24 (5.0)	0.34 (7.0)	0.43 (9.0)	0.58 (12.0)	0. 58 (14.0)
1988-1991	1814	0.11 (2.2)	0.09-0.13 (1.8-2.7)	<0.05 (<1.0)	<0.05 (<1.0)	0.07 (1.4)	0.12 (2.4)	0.19 (3.9)	0.31 (6.4)	0.41 (8.4)
Income level, low†		····								
1982-1984	2460	0.42 (8.8)	0.42-0.44 (8.6-9.1)	0.19 (4.0)	0.24 (5.0)	0.29 (6.0)	0.43 (9.0)	0.58 (12.0)	0.7 7 (16.0)	0.92 (19.0)
1988-1991	1664	0.16 (3.3)	0.13-0.19 (2.7-4.0)	<0.05 (<1.0)	0.06 (1.2)	0.10 (2.0)	0.17 (3.6)	0.28 (5.8)	0.43 (9.0)	0.54 (11.1)
Income level, midt										
1982-1964	2032	0.40 (8.3)	0.38-0.42 (7.9-8.7)	0.19 (4.0)	0.24 (5.0)	0.29 (6.0)	0.43 (9.0)	0. 58 (12.0)	0.77 (16.0)	0.87 (18.0)
1988-1991	1024	0.13 (2.6)	0.11-0:15 (2.2-3.1)	<0.05 (<1.0)	<0.05 (<1.0)	0.06	0.14 (2.9)	0.23 (4.8)	0.36 (7.5)	0.44 (9.2)
Income level, high† 1982-1984	674	0.39 (8.1)	0.37-0.39 (7.6-8.6)	0.19 (4.0)	0.24 (5.0)	0.29 (6.0)	0.39 (8.0)	0.53 (11.0)	0.72 (15.0)	0.82 (17.0
1988-1991	393	0.11 (2.3)	0.09-0.14 (1.8-2.9)	<0.05 (<1.0)	<0.05 (<1.0)	0.08	0.12 (2.5)	0.19 (4.0)	0.28 (5.7)	0.35 (7.3)

^{*}For each grouping, the geometric means from HHANES and NHANES III phase 1 are statistically different (P<.01). †Income level was defined by poverty-income ratio (PIR) categorized as low (0<PIR<1.30), mid (1.30≤PIR<3.00), and high (PIR≥3.00).

housing. The distribution of blood lead levels in the NHANES reflects exposure in the general population, whereas studies focusing on high-risk populations, such as persons living in older, deteriorating housing, may find a different blood lead distribution. Data from national housing surveys indicate that in 1980 about 24.2 million (30.3%) occupied houses in the United States were built before 1940 when lead-based paint was in common use. By 1989, this number had decreased by 3.4 million to 20.8 million (22.2%), suggesting that population exposure to lead-based paint may have decreased slightly.21.22 On the other hand. the continuing deterioration of leadbased paint in existing houses could increase the likelihood of exposure for persons in the 20.8 million households who remained in these older houses. On a population scale, it is not clear whether the net effect is an increase or decrease in exposure to lead-based paint.

The consistent decline in blood lead levels across broad population categories of age, sex, race/ethnicity, urban status, and income level most probably reflect changes in exposure to major population-wide lead sources. In addition, selected population groups within the United States are likely to have benefited from other changes in exposure, such as reductions in lead in community water supplies and reduction of lead emissions from local industry.

The public health impact of the observed decline in blood lead levels of the US population is dramatic, especially for children. The change in the proportion of children aged 1 to 5 years with blood lead levels 0.48 µmol/L (10 µg/dL) or greater was at least 70% for non-Hispanic whites, non-Hispanic blacks and Mexican Americans. Although the decline in blood lead levels is encouraging, the number of children with lead levels 0.48 µmol/L (10 µg/dL) or greater remains substantial and disproportionately higher for non-Hispanic black children (one in five children), as discussed in the accompanying article in this issue.

At least 99.8% of lead in gasoline has already been removed; and domestically produced cans are no longer lead soldered. Therefore, to achieve additional

Table 4.—Percentage of Mexican Americans Aged 4 to 74 Years at or Above Selected Blood Lead Level Cutoffs by Age, Sex, and Income Level: 1982 to 1984 (Hispanic Health and Nutrition Examination Survey) and 1988 to 1991 (Phase 1 of the Third National Health and Nutrition Examination Survey)

	3	Blood Leed Levels of Population Group, %							
	i No	≥1.45 µmol/L (≥30 µg/dL)	≥1.21 µmoVL (≥25 µg/dL)	≥0.97 µmoVL (≥20 µg/dL)	≥0.72 µmol/L (≥15 µg/dL)	≥0.48 µmol/L (≥10 µg/dL)	≥0.24 µmol/L (≥5 µg/dL)		
All persons 1982-1984	5682	0.4	1.3	3.6	12.8	41.2	91.2		
1988-1991	3611	0.0	0.2	0.4	1.5	5.9	28.9		
Ages 4-5 y 1982-1984	269	2.4	4.9	8.6	24.7	61.5	96.4		
1988-1991	349	0.0	0.0	0.0	0.1	4.9	32.7		
Ages 6-19 y 1982-1984	2331	0.3	0.5	2.0	9.0	35.8	90.1		
1988-1991	1188	0.0	0.1	0.4	0.9	4.5	23.8		
Ages 20-74 y 1982-1984	3082	0.3	1.5	4.2	14.1	42.9	91.5		
1988-1991	2074	0.0	0.2	0.4	1.9	6.6	31.0		
Maies 1962-1964	2638	0.6	2.1	6.2	21.1	58.4	96.8		
1988-1991	1797	0.0	0.2	0.5	2.2	8.7	40.5		
Females 1982-1984	3044	0.1	0.4	1.1	4.5	23.8	85.6		
1988-1991	1814	0.0	0.1	0.3	0.7	2.8	18.4		
income level, low ^e 1982-1984	2460	0.5	1.4	4.1	14.6	45.2	92.2		
1988-1991	1664	0.0	0.1	0.4	1.7	7.3	33.5		
income level, mid ^e 1982-1984	2032	0.4	1.3	3.4	11.4	38.0	90.1		
1986-1991	1024	0.0	0.0	0.3	1.1	4.2	23.6		
Income level, high* 1982-1984	674	0.2	0.8	2.7	10.0	35.8	91.2		
1988-1991	393	0.0	0.0	0.0	0.0	1.6	14.7		

thcome level was defined by poverty-income ratio (PIR) categorized as low (0<PIR≤1.30), mid (1.30≤PIR<3.00), and high (PIR≥3.00).

reductions in blood lead levels in the US population, sources other than lead in gasoline and lead in solder need to be

addressed further. The major remaining sources are lead in paint and lead that has already accumulated in dust and soil. Without efforts to reduce these exposures, population blood lead levels are unlikely to continue to decline.

1. Agency for Toxic Substances and Disease Registry. The Nature and Extent of Lead Poisoning in Children in the United States: A Report to Congress. Atlanta, Ga: US Dept of Health and Human Services, Public Health Service; 1988.

2. Mahaffey KR, Annest JL, Roberts J, Murphy RS National estimates of blood lead levels: United States 1976-1980. N Engl J Med. 1982;307:573-579. 3. Annest JL, Pirkle JL, Makuc D, Neese JW, Bayse DD, Kovar MG. Chronological trend in blood lead levels between 1976 and 1980. N Engl J Med. 1988; 308:1373-1377.

4. National Center for Health Statistics. Plan a operation of the second National Health and Nutrition Survey, 1976-80. Vital Health Stat 1. 1981; No. 15. US Dept of Health and Hussan Services Publication PHS 81-1317.

5. National Center for Health Statistics. Plan and operation of the Hispanic Health and Nutrition Examination Survey, 1982-94. Vital Health Stat 1. 1985; No. 19. US Dept of Health and Human Services publication PHS 85-1321.

6. National Center for Health Statistics, Execti TM, Massey JT, Waksburg J, Chu A, Maurer KR. Sample design: third National Health and Nutrition Examination Survey. Vital Health Stat 2. 1992; No. 113. US Dept of Health and Human Services publication PHS 92-1387.

7. National Center for Health Statistics, Anne JL, Mahaffey K. Blood lead levels for persons 6 months-74 years of age, United States, 1976-80. Vital Health Stat 11. 1984; No. 223. US Dept of Health and Human Services publication PHS 84-1683.

8. Carter-Pokras O, Pirkle JL, Chavez G, Gunter E. Blood lead levels of 4-11 year old Mexican-American, Puerto Rican, and Cuban children. Public Health Rep. 1990;105:388-398.

9. Brody DJ, Pirkle JL, Kramer RA, et al. Blood lead levels in the US population: phase 1 of the third National Health and Nutrition Examination Survey (NHANES III, 1988 to 1991). JAMA. 1994; 272:277-283.

10. Centers for Disease Control. Laboratory Procodures Used by the Clinical Chemistry Division, Centers for Disease Control, for the Second National Health and Nutrition Examination Survey (NHANES II) 1976-1980. Atlanta, Ga: Centers for Disease Control and Prevention; 1981.

11. Gunter EW, Miller DT. Laboratory Procedures Used by the Division of Environmental Health Laboratory Sciences, Center for Environmental Health, Centers for Disease Control, for the Hispanic Health and Nutrition Examination Survey (HHANES) 1982-84. Atlanta, Ga: Centers for Disse Control and Prevention; 1986.

12. Miller DT, Paschal DC, Gunter EW, Stroud PE, D'Angelo J. Determination of lead in blood using electrothermal atomisation atomic absorption spectrometry with a L'vov platform and matrix modifier. Analyst. 1987;112:1701-1704.

13. SAS Institute Inc. SAS Language: Version 6. Cary, NC: SAS Institute Inc; 1990.

14. Shah BV, Barnwell BG, Hunt PN, Lavange LM. SUDAAN User's Manual, Release 5.50. Research Triangle Park, NC: Research Triangle Institute; 1991.

15. Environmental Protection Agency. Quarterly

Summary of Lead Phasedown Reporting Data. Washington, DC: Office of Mobile Sources, Office of Air and Radiation, US Environmental Protection Agency; 1991.

16. Centers for Disease Control. Preventing Lead Poisoning in Young Children: A Statement by the Centers for Disease Control. Atlanta, Ga: US Dept of Health and Human Services, Public Health Service: 1991.

17. Environmental Protection Agency. Air Quality Criteria for Lead. Research Triangle Park, NC: Office of Health and Environmental Assessment; 1986. Environmental Protection Agency report EPA/600/8-83/028aF.

18. Can Manufacturers Institute. Food and Soft Drink Can Shipments. Washington, DC: Can Manufacturers Institute; 1992.

19. Bolger PM, Carrington CD, Capar SG, Adams MA. Reductions in dietary lead exposure in the United States. Chem Speciation Bioavailability. 1991:3:31-36.

29. Adams MA. FDA total diet study: dietary intakes of lead and other chemicals. Chem Speciation Bioavailability. 1991;3:37-41.

21. US Dept of Commerce, US Dept of Housing and Urban Development. Annual Housing Survey. 1980: Part A: General Housing Characteristics. Washington, DC: US Dept of Commerce; 1982. Current Housing Reports series H-150-80.

22. US Dept of Commerce, US Dept of Housing and Urban Development. American Housing Survey for the United States in 1989. Washington, DC: US Dept of Commerce; 1991. Current Housing Reports series H-150-89.